ON

A CASE OF

ACUTE ATROPHY OF THE KIDNEYS AND LIVER IN A PREGNANT WOMAN.

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In a paper which was published in this journal for October 1865, I drew attention to the fact that, in cases of acute yellow atrophy of the liver, the same changes may exist in the kidneys that occur in the hepatic organ, and endeavoured to show that in both the process depends upon a blood-poison. I have now to describe a case apparently of the same nature, in which the kidneys were primarily affected, the liver only secondarily, and to a slighter extent.

For an account of the symptoms I am indebted to Drs Graham Weir, M'Cowan, Mitchel, and Oman, who saw the patient during the few days of her illness.

M. M., aged 22, was of dissipated and abandoned habits, but always enjoyed good health till the commencement of this illness. In October she was far advanced in pregnancy, and on the 20th of that month, without known cause, she became suddenly ill, with symptoms which were referred to inflammation of the uterus. But as no satisfactory treatment was possible where she resided, and as she became rapidly worse, she was sent to the Maternity Hospital. She was admitted on Friday evening, 22d October. She was then very weak, and was vomiting stringy mucus, with streaks and clots of blood, and now and then matter like coffee-grounds. This continued, notwithstanding the employment of suitable remedies, until Sunday morning, October 24, when she began to sink rapidly. At that time premature labour was brought on by direction of Dr Graham Weir. She was delivered of a putrid child about 4 P.M., and died

at 8 o'clock the same evening. She was never unconscious, but was quite intelligent until within a few minutes of her death. She had also no convulsions, nor any distinct delirium. She passed urine several times after her admission, but it was in small quantity, and its qualities were not ascertained.

Autopsy forty hours after Death.—The body was well nourished. There was no dropsy. Decomposition was rapidly advancing. The pericardium was natural. The heart contained a moderately firm clot, but the blood generally was dark and fluid. The muscular substance of the heart was flabby, but the valves were healthy. Both lungs were congested throughout, particularly towards the base posteriorly. There were traces of old tubercle at the left apex. The liver was of natural size, soft, and flabby, but not friable; the outlines of its lobules were distinct. The spleen was pulpy. The kidneys were small, and weighed together $6\frac{3}{4}$ ounces. They were flabby, congested, and in the cortical substance there were numerous extravasations. That part was not increased, but rather diminished in size, and contained some yellowish-white deposit, which was rendered the more distinct by the surrounding congestion. The stomach contained some dark green matter, like altered blood; its mucous membrane was catarrhal, and was perforated by numerous minute ulcers. Some parts were deeply congested, and at others there were patches of extravasation. The upper part of the small intestine contained bile and altered blood. The coats throughout were normal. The large intestine was natural. The ovaries were natural. The uterus presented the ordinary appearance of one recently delivered, and was moderately contracted. The bladder contained almost no urine; its coats were normal. The head was not examined.

Microscopic examination of the Kidneys.—The whole organ was found to be altered. The tubules, both in the cortical substance and in the cones, were opaque, and appeared as if distended with fine dark injection. On examination with a higher power, the distention of the tubules was found to depend partly upon enlargement of the epithelial cells and partly upon free exudation, which occupied the cavity of the tube. The cells were in some parts opaque, of a brownish colour, and full of dense granular matter, which obscured the nuclei; in others, they contained fatty granules of large size, surrounded by numerous firm fatty molecules; in others, they were represented by groups of oil globules, enclosed by an indistinct cell wall, which broke down under very slight pressure. So great was the friability of these fatty cells, that when a scraping of the kidney was examined, the pressure of a light covering glass sufficed to break down the tube-casts to a fine molecular debris. In transverse sections of the tubules the changes were well seen; in some the epithelium enlarged, dense brown, filling up the lumen; in others, the opaque matter replaced by fat, the outlines of the cells remaining entire; in others, the cells broken down, and represented by groups of fat granules; others were entirely empty.

The condition of the stroma was also interesting; it was somewhat enlarged, and contained numerous fatty granules. Many of these were arranged in little groups and lines, and appeared as if situated in the connective tissue-corpuscles. The corpuscles were certainly somewhat enlarged, from which it appeared that the lesion was not confined to the tubules.

From the fact that, although every tubule which was seen was distended, the cortical substance was diminished rather than increased, and the organs were below their average weight, it is evident that considerable wasting of the organ must have taken place, and this we can well understand when we think of the rapid fatty disintegration which was going on. The distinct diminution of bulk of the organs, and the rapidity with which it was taking place, seem to warrant the application of the name, "acute atrophy."

The very striking similarity between the conditions just described and those accompanying acute yellow atrophy of the liver cannot be overlooked, whether we regard the general appearance of the organ or its microscopic characters. The whole series of changes, from the exudative infiltration into the complete disintegration of the cells, was identical with what was found in the case recorded in this journal for last October.

Microscopic examination of the Liver.—Under a low power the hepatic cells were seen to be unusually opaque, while towards the margin of each lobule there was a dark zone of fatty degeneration, and outside of that—*i.e.*, at the point of contact of neighbouring lobules—there was a clear space in which no cell was visible, only a little granular debris. Under a higher power, the cells were found to present exactly the characters of those met with in acute yellow atrophy—that is, some were enlarged, opaque, full of dense granular matter; others were fatty; others were in an advanced state of fatty degeneration, extremely friable, easily broken down. These different stages were also distributed in the lobules, just as was described in my former case—the swollen infiltrated cells being in the centre, surrounded by those in a stage of degeneration, these again surrounded by the space in which no cell element remained. There was no breaking down of the stroma of the organ, nor any marked bile-staining of the cells, nor retention of bile in the ducts.

It may be suggested that this change was an ordinary fatty degeneration, occurring in its usual seat, towards the margin of the lobule, and not at all to be wondered at, considering the character and habits of the patient. But I think it was something more,—firstly, because the appearance of the fatty cells was unlike what we ordinarily see; secondly, because of the peculiar opacity of the cells in the centres of the lobules; thirdly, because fatty degeneration never leads to destruction of the cells, as was seen in the periphery of the lobules, while in all these points the appearances are characteristic of the acute yellow atrophy.

It appears to me that we are entitled to regard this case as an example of a disease essentially identical with acute yellow atrophy, for the following reasons :—

1*st*. The pathological appearances in the kidneys and liver were the same as those met with in acute yellow atrophy.

2*d*. The leading symptoms of this case; the peculiar malaise; the sickness and vomiting of blood; the rapid sinking; the congestion of the lungs,—are among the chief of those looked for in the other disease.

3*d*. The circumstances of the patient. She was advanced in pregnancy, was leading an exposed and wretched life,—two circumstances very commonly associated with the other affection.

From these considerations, my view seems to be well established; at the same time, it is to be noticed that three frequent symptoms were entirely awanting, viz., delirium passing into coma, icterus, and diminution of the volume of the liver.

The absence of coma and convulsions seems unaccountable from the condition of the kidneys; for one would have expected well-marked uræmic symptoms. The bladder was empty, and the kidneys were obviously incapable of secretion. But the symptoms of neither the acute nor chronic form of uræmia were present. This fact can, I think, only be explained on the hypothesis, which is consistent with the other facts of the case, that the patient died before the uræmic symptoms had time to be developed.

The icterus and the diminution of the volume of the liver often occur late in the case, sometimes not at all; and in this instance the absence of diminution was well explained by the fact that the disease was not far advanced in the liver, and this circumstance also accounted for the absence of icterus.

In regard, again, to the symptoms which were present, this case seems to teach an important lesson; for the hæmatemesis, which is so frequent a symptom in acute yellow atrophy, occurred here, although the liver was comparatively little diseased; thus showing that hæmatemesis is not dependent on the hepatic affection. May it not be the result of a blood-poison?

If it be admitted that this case is of the same nature as acute yellow atrophy, it certainly gives the *coup de grace* to the theory which would make the renal affection secondary to the hepatic.

This case remarkably confirms the opinion that acute yellow atrophy is a blood disease. The general appearance of the body; the marked hypostatic congestion and rapid decomposition; the softness of the spleen; the darkness of the blood and its imperfect coagulation; the occurrence of identical morbid conditions in the liver and kidneys; and the peculiar nature of the process in both organs, differing, as it does, from ordinary inflammation and from ordinary fatty degeneration, and the peculiar circumstances under

which it occurred, point strongly to some cause operating within the blood.

The case, then, supplies an additional point in the history of acute yellow atrophy, and decidedly supports the view of its being dependent on a blood-poison.

The only similar cases that I have found are three, which were published by Rokitansky in 1859, under the name of fatal steatosis of the liver and kidneys.[1] I shall not detail his cases, but merely mention certain general facts in regard to them. All were females, unmarried, aged 23, 38, and 8 years respectively. Two had been in depressed spirits. All were stout, well-nourished, and died after about a week's illness, during which the leading symptoms were—fever, headache, vomiting of dark matters, convulsions, and coma; in two there was slight icterus. On post-mortem examination, ecchymoses were found in many of the internal organs. The livers were large, pale, flabby, and fatty; the kidneys were also large, fatty, congested, and mottled with extravasation of blood; the urinary bladders contained almost no urine; in two, the stomach and intestines contained altered blood. Microscopic examination showed in all three cases extreme fatty degeneration of the liver and kidneys.

Rokitansky distinctly recognises a poisoned condition of the blood, which, however, he regards as a consequence, not a cause, of the hepatic and renal affections; and, in accordance with a favourite view, he regards the renal affection as secondary to the hepatic. He refers the fatty condition of the liver to that state of the system in which an excess of fat is present and a tendency to fatty deposition exists, and having excluded, with regard to the kidneys, the theory that it might depend upon Bright's disease, he concludes by saying, "There can be little doubt that the steatosis of the kidney was secondary to the primary steatosis of the liver; just as little can we doubt—from the diminution of urine consequent upon kidney disease on the one hand, and the slight degree of cholæmia on the other hand—that the sudden development of the fatal symptoms depended upon uræmia. It would accordingly appear that fatty degeneration of the liver may occur in an individual inclined to excessive fatty deposit, and to this, sooner or later, a fatty condition of the kidney may be superadded, both of which, gradually and unobserved, may attain such a degree that secretion of bile and urine is suspended, and death supervenes suddenly, after the appearance of slight icterus, by uræmia, and an hæmorrhagic decomposition of the blood."

Notwithstanding the profound respect to which every opinion of Rokitansky is entitled, I would venture to suggest that the cases are capable of another interpretation, especially when examined by the light of the remarkable case which I have just described. It is

[1] Ueber lethale Leber- und Nieren Steatose, von Prof. Rokitansky. Zeitschrift der k. k. Gesellschaft der Aerzte zu Wien, Aug. 1859.

very difficult to believe that so general and important an affection of the liver and kidneys should have gradually advanced, and yet for long led to no symptom; still more is it difficult to imagine how so insidious an affection should have suddenly produced such violent symptoms, how the uræmia should have been unaccompanied by any trace of dropsy, or should have led to a hæmorrhagic condition of the blood; and, on the other hand, all the symptoms correspond closely with those of acute yellow atrophy, and many of them with that peculiar variety of the disease which I have described. These cases, indeed, seem to constitute a connecting link between the affection in its ordinary form, in which the liver is mainly involved, and this peculiar variety in which the kidneys are the primary seat of disease.

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